handling and blocking off of protruding intestines. Moreover, it will provide the safe and efficient drainage demanded in most of these conditions. An anterior incision should be reserved for injuries which presumably involve the intraperitoneal organs; even in these cases a supplementary lumbar incision for drainage may be indicated, particularly if a nephrectomy is not performed.

Treatment of the Injured Kidney. Nephrectomy should be reserved for the cases in which the integrity of the kidney cannot be preserved, and it is obvious that hemorrhage cannot be effectually stopped or prevented otherwise, or the outflow of the urine into the ureter cannot be effectually restored. In the event of doubt arising regarding the integrity of the other kidney, nephrectomy may be deferred until sufficient information is obtained. Meanwhile, the injured kidney should be attended to, the peritoneum if torn should be sutured or packed, lacerations sewed if advisable, the pelvis drained, and the whole or part of the wound packed and drained efficiently. Where nephrectomy is not required, suture or packing with drainage will suffice. How much more efficient suture rather than packing a lacerated area will prove is to me an open question. I think not much time should be lost in performing it, and it should perhaps be reserved for cases in which packing may less efficiently check bleeding. The main indication is to provide free drainage which will minimize the disastrous secondary effects of injury and extravasation.

This paper is written to call attention to the fact that rupture of the kidney in children is probably commoner than generally estimated. That the lesion is frequently severe, consisting of a complete division of the kidney into unequal halves. That shock and other symptoms may be slight and out of proportion to the gravity of the lesion. That operative interference should be more freely employed and gives good results.

THE SO-CALLED "MIXED" TUMORS OF THE SALIVARY GLANDS.

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The mixed tumors of the salivary glands were carefully reviewed by Wood in 1904. His article gives a detailed report of his observations on the pathology of 42 cases and contains a clear *resume* and fairly complete bibliography of the pathologically significant literature to that date. His conclusions in brief are as follows:

"1. There is a group of extremely complicated tumors occurring in the facial region which contain elements from both epi- and mesoblast in most intricate relationship to each other.

"2. The complicated structure of the stroma, containing as it does elements such as embryonic connective tissue, cartilage, bone, fat, and lymphoid tissue, and very rarely striated muscle is explained most easily by the assumption of an embryonic misplacement of mesoblast.

"3. The structure of the parenchyma is so slightly characteristic in morphology that its epithelial nature in all cases can only be considered as probable; yet in about 24 per cent. of the tumors examined the presence of epithelium is undoubted. The form and relationships of the cells of the parenchyma do not furnish sufficient data to justify these cells in being regarded as of epithelial origin.

"4. The theory of early embryonic displacement of epiblastic tissue during the process of formation of the parotid and submaxillary gland and the branchial arches may account for many of the morphological peculiarities of the cells of these tumors, especially the lack of any typical features which we associate with epithelium. The same conditions may be seen in the epithelial cells of the congenital moles in which the epithelium is with difficulty distinguished from connective-tissue cells, owing to its close connection with the stroma of the tumors and its undifferentiated type.

"5. The mixed tumors of the salivary gland run a clinical course strikingly different from the sarcomas and carcinomas in that they are slow growing and generally benign. The regional lymph nodes are not invaded and recurrences are likely to remain local in a considerable proportion of the cases."

Since the publication of Wood's article an extensive literature has grown up. A bibliography thereof is appended. The following papers may be briefly noted as of pathological interest: Verhoeff reports 5 cases of tumors of the lacrymal gland and discusses the various theories of the formation of these tumors and of the mixed tumors of the salivary gland. His conclusions are as follows:

"The mixed tumors of the lacrymal and salivary gland (so-called endotheliomas) are essentially epiblastic in origin. The stroma of these tumors is not derived from mesoblastic cells misplaced from other structures, but is probably produced by an atypical development of cells which ordinarily would have gone to form part of the stroma of the normal gland. Owing to their situation the tumors of the lacrymal gland are not only dangerous to sight, but are more dangerous to life than those of the salivary gland and should be extirpated as soon as possible."

Keen observed one of the largest mixed tumors of the parotid reported, the specimen weighing seven pounds.

Ehrich reports his observations on the pathology of 33 tumors of the salivary gland. He gives a detailed report of the cases, both clinically and pathologically, and a lengthy discussion of the literature.

Martini, who has studied 6 cases, reviews the literature at great length and discusses the various hypotheses of origin of the tumors. He noted in his cases that in the peripheral parts of the tumors the stroma consisted of a more or less strongly developed framework of fibrous or reticular structure which was directly united with the capsule and that the same in advancing toward the centre of the tumors changed its structure, assuming the appearance of thick and tough or soft and loose connective tissue or a myxomatous or cartilaginous connective tissue. . . . No sharp limit existed between the various kinds of connective tissue. . . . The endotheliomatous formations were more luxuriant in those zones where the stroma had a fibrous structure. He concludes: "(1) The presence of fibrous connective tissue in the youngest parts of a tumor and of myxomatous and cartilaginous tissue in the oldest parts of the same indicate that these varieties of the connective tissue present the product of a metaplastic process of the fibrous stroma and not the product of processes developing at the cost of *germi-aberrantes* or other mesenchymal elements. (2) Besides the different phases of evolution of the neoplastic cells and their slow or rapid growth one observes the various phases of alteration and transformation of the stroma. . . . The question of the significance and origin of the myxomatous and cartilaginous tissue in the mixed tumors remains still undecided."

Martini was unable to identify the presence of true gland tissue in his mixed tumors. He asserts that the "acini" are without a limiting membrane and that the supposed epithelial cells are intimately related to the stroma. He thinks he can trace the origin of the tubules and cell formations from proliferated endothelium in the lymph vessels.

Massabuau reports on 4 mixed tumors of the parotid, 1 of the submaxillary gland and 1 of the velum palati. He discusses at length the literature and various theories of development, but finally concludes, "the theory which explains in a perfect manner the structure of these mixed tumors of the salivary gland is that which explains their development from ectomesodermic rests of the embryonic bud which was destined to the formation of the glands themselves. (1) It is the one important fact in the development of the mixed tumors on the border of glandular organs. (2) It explains the new formation of the flat epithelium as well as of the normal and edematous glandular epithelium. . . . (3) It explains perfectly the *intrication* of the two orders of endothelial proliferation. . . . (4) There is no need of invoking, as in the endothelial theory, a cellular metaplasia for explaining the development of the polymorphous connective stroma of these tumors."

Speese reports his study of 9 cases, 8 of which were tumors of the parotid gland and 1 sublingual.

Fick reports a careful study of 2 cases, which he thinks do not support the hypothesis that tumors are endotheliomas.

Cordier reports in detail 1 case.

Von Hansemann has recently studied one enchondroma of the parotid in which he thinks he can without doubt trace the development of the parts from lymphangioma. He says, "I consider in fact this tumor a lucky finding which makes it possible to advance the sureness of the endothelial nature of the so-called parotid enchondromas. That lymph angiomas are present in the parotid has been long established. That, up to this time, such characteristic stages as are present in the tumor in question were not found is because these tumors were only seldom studied in the initial stages."

Sertoli makes a careful study of 4 cases and assumes that though the tumors may be of endothelial nature this is not opposed to the theory of their development from *germi aberrantes*.

The object of the present paper is to place on record the results of the analytic study of 56 mixed tumors of the salivary glands, which have been removed at operation at St. Mary's Hospital (Mayo Clinic), from January 1, 1905 to April 10, 1911.

The following tables give the sex and age incidence of onset, duration, and period of operation of the cases of "mixed" tumors of the salivary glands which we have studied:

TABLE I.—Sex and Age Incidence at Onset of Symptoms.

Decades:	2d	3d	4th	5th	6th	7th	Total.
Cases, males	3	10	10	3	3	1	30
Cases, females	6	5	6	6	3	..	26
Total	9	15	16	10	6	1	56

TABLE II.—Time from Onset of Tumor to Date of Operation.

Time:	1 mo.	4 mo.	8 mo.	9 mo.	1 yr.	2 yr.	3 yr.	4 yr.	5 yr.	6 yr.	7 yr.	8 yr.	10 yr.	13 yr.	15 yr.	20 yr.	25 yr.	27 yr.	30 yr.	44 yr.	Total.
Cases, males .	1	1	1	1	4	5	1	.	1	3	1	1	3	1	2	1	.	1	1	.	30
Cases, females .	1	.	.	.	5	4	3	2	2	4	1	2	.	.	.	.	1	.	.	.	26
Total . .	2	1	1	1	9	9	4	2	3	7	2	3	3	1	2	1	1	1	1	1	56

TABLE III.—Sex and Age Incidence at Time of Operation.

Decades:	2d	3d	4th	5th	6th	7th	8th	Total.
Cases, males	2	5	8	8	3	3	1	30
Cases, females	3	4	7	5	4	3	..	26
Total	5	9	15	13	7	6	1	56

From these tables it will be seen that the sexes are almost equally divided, but with a slight preponderance of males. From Table I above it will be seen that the age at onset in more than two-thirds of the cases is under forty years, while in one-half the cases it falls between twenty and forty years. These figures agree, on the whole, with those of other observers. For example, of Wood's 42 tumors, 4 appeared in the first decade, 1 in the second, 13 in the third, 8 in the fourth, 9 in the fifth, 4 in the sixth, and 3 in the seventh. Thus almost two-thirds of his cases also began before the fortieth year. When the figures for Wood's series and ours are added, the percentages by decades at time of onset are approximately as follows: First decade, 4 per cent; second decade, 10 per cent.; third decade, 28 per cent.; fourth decade, 24 per cent.; fifth decade, 19 per cent.; sixth decade, 10 per cent.; seventh decade, 4 per cent. These percentages are considerably advanced over those of Kaufmann and Küttner, who describe the majority of these growths as beginning in the second decade. Looked at broadly, the salient point of these figures is that 66 per cent. of the tumors began before forty years of age and 85 per cent. of them before fifty years of age.

Some interest attaches to the length of the period between the onset of the tumor and the time of surgical operation. As will be seen from Table II, this varies from one month to forty-four years, with a large preponderance of the cases running from one to ten years, and an average of six years and four months. The average of Wood's cases for the same period was eight years and nine months. When the figures for his 42 cases and our 57 cases are combined (total 99 cases) they give an average duration of symptoms prior to operation of seven years and four months. The obvious lesson from this is that these tumors do not cause sufficient inconvenience to the patients to make them seek operation early. In only 14 of our cases was there a history of pain. It is difficult to say, whether, if the patients were seen earlier, it would have a material effect on the final results of the operation, since, as will be noted later, the tumors which are carried longest without giving unpleasant symptoms are those which are apparently benign. The age at operation, as shown in Table III, indicates that the majority of these patients are in a favorable period of resistance.

Acute Parotitis. Certain observers have attempted to trace a relationship between mumps and "mixed" tumors of the salivary glands. Of the 21 of our patients with tumors of the parotid who could give a definite history concerning mumps, 14 (66 per cent.) were sure they had never had the disease. In other words, there seems to be no etiological relationship established by the statistics of mumps and "mixed" tumors of the parotid.

Location and Gross Appearance. Of our 56 tumors, 50 were in the parotid and 6 in the submaxillary glands. Thirty were on the right side and 26 were on the left side. Grossly, the tumors may be divided into four groups: (1) Hard, fibrous masses with little cartilage and with little parenchyma (see Fig. 1); in this group were 15 cases. (2) Very hard tumors containing large cartil-

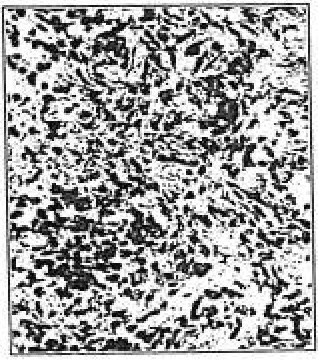

FIG. 1.—Case XIII. Group 1. Dense fibrous tumor.

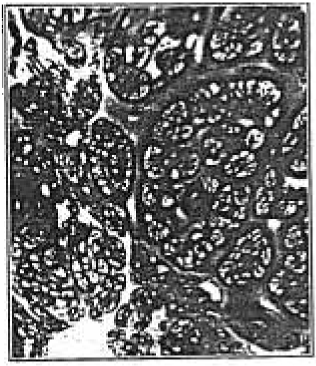

FIG. 2.—Case X. Group 2. Area showing cartilage.

age masses (see Fig. 2); in this group were 17 cases. (3) Soft, more or less sarcoma-like tumors with relatively small amount of fibrous connective tissue and a relative large amount of parenchyma cells (see Figs. 3 and 4); in this group were 20 tumors. (4) Tumors grossly resembling carcinoma; in this group were 4 tumors.

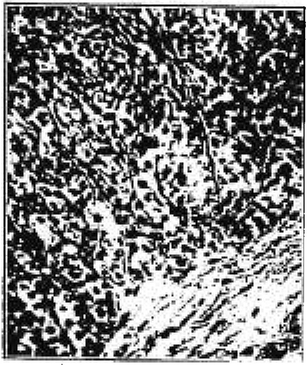

Fig. 3.—Case XVIII. Group 3. Loose parenchyma in cordons.

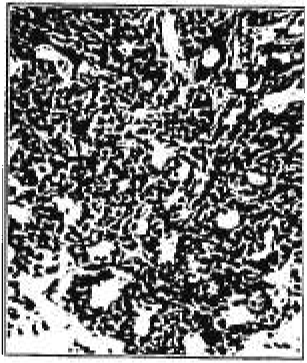

Fig. 4.—Case XXXV. Group 3. Parenchyma showing tubules.

Histology; Stroma. Thirty-three of our cases showed marked increase of the fibrous connective tissue; 17 of these showed cartilage formation. In all of the cases in which the parenchymatous element was in marked preponderance the connective tissue appeared to be of distinct embryonic type. This connective-tissue type of embryonic cells was also present in some areas of the more fibrous tumors. In 35 of the cases a more or less distinctly myxomatous tissue was present (see Fig. 5). In many tumors was present a transparent, hyaline structure. It was noted that the cartilaginous and myxomatous elements were apt to be near the centre, that is, older portions of the tumors, rather than near the advancing borders where the simple fibrous connective tissue and parenchyma were greatly in predominence or constituted the whole of the tissue. This, of course, suggests a metaplastic origin of the cartilage, hyaline, and mucoid structures.

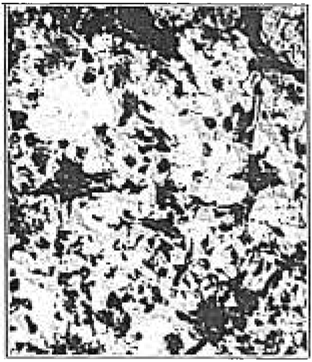

FIG. 5.—Case XVI. Group 1. Myxomatous change

Bloodvessels. In 24 cases more or less endarteritis was present. These were about equally distributed throughout the four groups as above classified from their gross appearance. In these cases the proliferation within the vessels did not seem to us as sufficient in amount or of such a type as to suggest tumor formation within them. On the contrary, the changes appeared to be rather the result of chronic irritation.

Parenchyma. In Group 1, that is, the hard, fibrous tumors, the parenchyma cells are usually found in small, distorted groups, which are frequently interpreted as the result of endothelial proliferation. The same is true in certain areas in Group 2, namely, the tumors with a marked cartilaginous element. Less rarely do such cell elements occur in the tumors of Group 3, those with loose, abundant parenchyma, and then only in small areas. On the other hand, in these parenchymatous tumors the cells of the advancing neoplasms are more frequently arranged in cordons (see Fig. 3) or tubules (see Fig. 4) which strongly suggest the normal histology of the gland. A careful examination of the flattened and distorted cell groups, so frequently interpreted as "endotheliomas," leads one to the belief that they are either (1) adult epithelium which has been flattened and distorted by pressure, or (2) proliferating embryonic transitional mesothelium which has never attained adult type. There is a direct relationship between the amount of parenchyma and the rate of development and malignancy of the tumors.

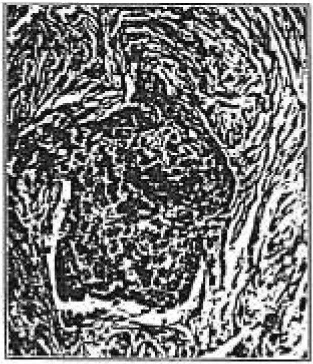

FIG. 6.—Case XV. Group 4. Mass of epithelium.

In many instances the variation in morphology in the parenchyma cells is so far retrogressive that it approaches the true spindle-celled sarcoma type. In some cases there is such a preponderance of cells of the aberrant, epithelial type (see Fig. 6) that one hesitates whether or not to diagnosticate such tumors as carcinomas. We have, however, included within our series only those cases of salivary gland tumors which show a newgrowth of two or more of the types of fibrous connective tissue, cartilage, sarcoma cells or adult epithelial cells.

All degrees of adhesion to and shrinkage away from the alveolar walls by the lining parenchyma have been found. This point which has been made so much of by Volkmann we consider as having little diagnostic value.

Etiology. The origin and development of the so-called "mixed" tumors of the salivary glands must be considered in relation to the origin and development of other "mixed" tumors. These have recently received considerable study. Attention may be called to the articles by Stoerk and by the present writers attacking the Grawitzian hypothesis of the so-called renal "hypernephromas," and those by Ewing and Stoppato on the teratomas of the testicle.

There is, however, little to add to the discussion of the derivation of these tumors as given by Wood. Perhaps the matter of most interest related to their development is the growing belief of embryologists that the parotid gland takes its rise much earlier in the embryo than was formerly supposed. It now seems to be somewhat definitely settled that the *anlage* of the parotid appears about the fourth week instead of the eighth week of embryonic life as was formerly taught. At this period the mesoblastic tissues through which the gland bud forces itself are much less differentiated than they are in the eighth week. These facts are, therefore, in better accord with the hypothesis that the "mixed" tumors of the parotid are derived from areas of imperfectly developed mesoblastic tissue than was the former teaching of the later embryonic development of the parotid gland. So far as our personal observation goes, it would seem to us that the weight of evidence is in favor of the theory that these tumors are the result of proliferation of mesoblastic tissue perhaps displaced early in the embryonic development of the glands or which for some reason has failed to attain adult characteristics.

Results of Operation. Thirteen of our cases had been operated on elsewhere before they appeared at this clinic. These, therefore, represent cases of recurrence. Of the 42 cases which have been heard from since being operated on here, 9 have had a recurrence of the disease. Five of these were cases in which the neoplasm was of the loose, markedly sarcomatous type, and 4, cases in which the original growth was of the hard, fibrous type. Five of these 9 cases have died. Of these 5, 2 cases had tumors with a large epithelial element and in 3 the largest element of the tumors was of sarcoma type. All of these cases have died away from the clinic and no opportunities have been afforded to make postmortem examinations.

As has been pointed out by Judd, the recurrences are caused mostly by the difficulty of removing all portions of the tumor and yet preserving the fibers of the seventh nerve. When a tumor is already sarcomatous or carcinomatous at the time of operation the chances for recurrences are materially increased.

Mixed Tumors of Parotid Glands.

Group, Case No.	Registered No.	Sex.	Age.	Duration of symptoms.	Previous operation.	Date of our first operation.	Subsequent history.	Side affected.	Previous parotitis.	Pain.	Rate of tumor growth: Rapid = R, Slow = S.	Macroscopic type of tumor.	Vessels: increase = +; decrease = —; endarteritis = E.	Stroma: increase = +; decrease = —; hyalin = H.	Cartilage.	Cell groups: alveoli = A; solid masses = SM.	Cells: cuboidal = C, flat = F; spindle = S, small round = SR.	Mitotic figures.	Myxomatous changes.	Calcification.
1	P. 2082	M	20	8 yr.	......	1905 Jan. 3	OK, 5 yr. 6 mo.	R	?	—	S	1	+	+H	—	—	C, S, SR	—	—	—
2	G. 4541	F	44	8 yr.	......	Mar. 27	OK, 4 yr.	R	?	+	R	1	—	+	—	—	S	—	—	—
3	G. 4758½	M	50	1 yr.	......	May 10	O. K., 3 yr.	L	—	—	S	1	—	+	—	—	S	+	—	—
4	P. 2691	F	37	7 yr.	......	May 25	O. K., 5 yr.	L	+	—	S	1	—E	+H	—	SM	F, SR	+	+	—
5	J. 812½	F	44	1 yr.	......	July 10	Rec., 5 yr.	L	?	+	S	3	+	—	—	SM	C, S	+	+	—
6	3043	M	33	2 yr.	......	July 20	Died 1 yr. 3 mo.	L	?	+	R	3	+	—	—	SM	C, F	+	—	—
7	S. 302	F	34	1 yr.	......	July 25	Rec., 2 yr.	L	?	+	R	3	+	—	—	SM	SR	+	+	—
8	Mrs. J. W.	F	57	8 yr.	......	1906 Mar. 7	O. K., 4 yr. 3 mo.	R	?	—	S	2	—	+H	+	—	C, F, S	+	+	—
9	J. 1597	F	47	5 yr.	......	June 23	O. K., 4 yr.	R	—	+	S	1	—E	+H	—	SM	F	—	+	—
10	Miss A. S.	F	20	3 mo.	......	July 25	O. K., 4 yr.	L	—	—	S	2	—	+H	+	SM	C, F	—	+	—
11	Pl. Bk., 14	F	38	2 yr.	......	1907 Mar. 18	O. K., 3 yr. 4 mo.	R	?	:	S	1	—E	+H	—	—	S	—	+	—
12	P. 6922	M	50	2 yr.	......	April 10	O. K., 3 yr. 2 mo.	L	+	—	S	2	—E	+H	+	SM	F, S	+	—	+
13	Be., 61	F	56	7 yr.	......	May 22	O. K., 3 yr.	R	?	—	S	1	—E	+H	—	SM	C, F, S	—	+	+
14	Gif., 1704	M	19	1 yr.	......	May 22	Rec. 1 yr. 6 mo.	R	?	—	S	3	+	—	—	SM	C, S	+	—	—
15	J. 2673	M	28	1 yr.	......	May 28	Rec. 3 yr. 4 mo.	L	?	—	S	3(4)	—	+H	—	SM	C, F	+	—	—
16	Dr. A. M. J.	M	37	2 yr.	......	June 10	O. K., 3 yr.	L	—	—	S	1	—	+H	—	—	S	—	+	+
17	Bk. or M6	F	30	2 yr.	......	June 11	......	L	?	—	S	2	—E	+H	+	—	C, F	—	+	+
18	J. 2738	M	47	10 yr.	6 yr.	June 15	O. K., 3 yr.	L	+	+	S	3	+	+H	—	A, SM	C, F, S	+	—	—
19	J. 2830	M	31	12 yr.	......	July 15	Rec., 3 yr.	R	+	+	S	3	+	+H	—	SM	C, F	—	+	—
20	Mr. C. J. F.	M	37	10 yr.	......	Aug. 7	O. K., 3 yr.	L	±	—	S	2	+E	+H	+	A	C, F	—	+	+
21	A1712	M	70	30 yr.	Inf.	Sept. 24	Rec., 6 wk.	R	?	+	S	4	—E	+H	—	—	C, F	—	—	+
22	A3042	F	20	1 yr.	......	Nov. 23	......	L	?	—	3 mo. R R	3	—E	—	—	A, SM	C, F	+	—	—
23	A5387	F	25	1 yr.	......	1908 Jan. 11	O. K., 2 yr.	L	+	+	S	1	—E	+H	—	SM	C, F	+	—	—
24	A7340	F	27	5 yr.	Op., 3 yr. 6 mo. Rec., 1 yr. 6 mo.	Mar. 7	Rec., 2 yr.	R	?	+	R	1	—	+	—	—	F, S	—	+	—
25	A11599	M	43	15 yr.	14 yr.	July 22	O. K., 2 yr.	R	?	+	S	2	+E	+H	+	SM	C, F	+	+	+
26	A183307	F	18	6 yr.	......	Sept. 10	O. K., 2 yr.	R	—	—	S	3	—	—	—	A, SM	C, S, SR	+	+	—

27	A12357	F	34	2 yr.	......	July 27	?	L	—	+	S	2	—E	+H	+	SM	F, S	—	+	+
28	A5175	F	31	6 yr.	......	Jan. 6	Rec., 1 and 2 yr.	L	—	+	R	3	—E	+H	—	A, SM	C, F	+	+	—
29	A18358	M	61	4 mo.	......	Dec. 28	Died 1 mo. later	L	?	+	R	4	—	—	—	SM	F, C	+	+	—
30	A4080	M	38	7 yr.	......	1000 Feb. 6	O. K., 1 yr. 6 mo.	L	?	—	S	2	—E	+H	+	A, SM	C, F	+	+	+
31	A22140	F	50	7 yr.	......	April 6	O. K., 1 yr. 2 mo.	R	+	+	S	1	—E	+H	—	A	SR	+	+	—
32	A21697	F	34	8 yr.	Op. 1 yr. ago	Mar. 27	Rec., 1 yr.	L	?	+	S	3	—	+H	—	SM	F, C	+	—	—
33	A22075	F	62	2½ yr. 6 mo.	......	April 21	O. K., 3 yr. 4 mo.	R	..	..	S	2	—E	+H	+	A, SM	F, S, SR	—	+	—
34	A26098	F	24	0 yr.	Op., 3 yr. Rec., 6 mo.	Oct. 14	Rec., 6 yr. Died 1 yr. 2 mo.	L	?	+	R	3	+	—	—	SM	F, C	+	—	—
35	A31134	F	43	3 yr.	......	Nov. 13	O. K., 1 yr.	R	?	+	S	3	—E	+H	—	SM	F, S	+	—	—
36	A33808	M	27	3 yr.	......	1910 Feb. 9	O. K., 4 mo.	R	?	+	S	3	—	+H	—	SM	F, C	+	—	—
37	A34574	M	30	0 yr.	......	Feb. 25	O. K., 4 mo.	R	?	—	S	2	+E	+H	+	A, SM	C	+	+	—
38	A37071	F	54	4½ yr. 0 mo.	......	April 3	O. K., 2 mo.	L	?	—	S	3	+	+H	—	SM	F, S	+	+	—
39	A37735	M	71	1 yr.	10 mo. ago	June 10	Died 3 mo.	L	?	+	R	3	—	—	—	SM	S	+	—	—
40	A38746	F	62	25 yr.	25 yr. 20 yr. 2 yr.	June 14	Rec. O. K., 3 mo.	R	—	+	R	2	—	+H	+	A, SM	F	—	+	+
41	A39230	M	55	20 yr.	......	June 23	......	R	?	—	R	3	—	—	—	A, SM	F	+	—	—
42	A39803	M	57	27 yr.	27 yr. 17 yr.	July 8	......	L	?	+	S	3	—	—	—	A, SM	C, F	+	+	—
43	A40330	M	25	5 yr.	......	July 16	Rec. after 4 mo.	R	—	+	S	3	+	—	—	A, SM	C, F	+	+	—
44	A14953	M	39	2 yr.	......	Aug. 22	......	R	?	+	S	2	—	+H	+	A	C, SR	+	+	—
45	A42737	M	42	2 yr.	......	Sept. 2	......	L	?	—	S	2	—	+H	+	A	F, C	—	+	—
46	A28505	F	58	1 yr.	......	Oct. 1	......	R	?	—	S	2	—	+H	+	A	C, F	—	+	—
47	A44048	M	60	8 mo.	6 mo. ago	Oct. 25	......	L	?	—	R	3	+	—	—	A, SM	C, F, SR	+	+	—
48	A46070	M	41	13 yr.	2 rec.	Dec. 12	......	R	?	—	R	1	—	+H	—	A	C, SR	+	+	—
49	A48731	F	24	4 yr.	......	1911 Feb. 7	......	R	?	—	S	1	—	—	—	—	C, SR	—	+	—
50	A51433	F	64	44 yr.	......	April 6	......	R	?	—	S	4	—	—	—	SM	F, C	+	+	—

MIXED TUMORS OF SUBMAXILLARY GLANDS.

1	P. 2403	M	39	6 yr.	......	1905 Apr. 8	O. K., 5 yr. 2 mo.	L	?	—	S	2	—E	+H	+	SM	C	+	+	—
2	G. 5570	F	46	1 mo.	......	Oct. 16	O. K., 4 yr. 8 mo.	R	—	+	R	4	—	+	—	A, SM	C, F, S	—	+	—
3	J. 1255	M	61	9 mo.	......	1906 Mar. 27	O. K., 4 yr.	L	—	—	R	1	+	+	—	—	S, SR	—	+	—
4	M. 2552	M	16	6 yr.	......	June 5	......	R and L	—	—	S	2	—E	+H	+	SM	C, S, SR	—	+	+
5	A21940	M	49	15 yr.	......	1909 Mar. 31	......	R	—	+	S	2	—E	+H	+	SM	C, S	+	+	+
6	A31575	M	40	1 mo.	......	Dec. 1	......	R	+	+	R	1	+E	+	—	—	S	—	+	—

Summary. 1. The present study covers 50 "mixed" tumors of the parotid and 6 "mixed" tumors of the submaxillary glands.

2. Most of the "mixed" tumors of the salivary glands arise in young adults.

3. Most of the tumors cause little inconvenience early and surgical relief is not sought until an average period of about seven years has passed.

4. Our cases show no etiological relationship between the tumors and acute parotitis.

5. The tumors which grossly are hard, consisting of fibrous connective tissue or cartilage are proliferating at their periphery only and are usually benign.

6. The tumors which grossly are soft, consisting largely of parenchyma of types varying from adult to embryonic are those among which are found the malignant tumors.

7. Connective-tissue and parenchymatous elements both tend to revert to embryonic type.

8. There is little evidence that these tumors arise from proliferating adult epithelium, or endothelium.

9. There is considerable evidence to support the theory that these tumors are mesotheliomas of embryonic origin.

10. After fairly complete operative removal few of the tumors recur.

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DUODENAL ALIMENTATION.[1]

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WHEN Einhorn first described his method of duodenal alimentation about a year and a half ago, I realized its possibilities and began using it in certain selected cases. After a considerable

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[1] Read before the New York Academy of Medicine, February 15, 1912.